BELL'S PALSY TREATED WITH CORTISONE

Review of Literature and Report of Cases

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Bell's palsy was first treated with cortisone in 1951, Rothendler\textsuperscript{1} reporting the first case. Cortisone therapy was begun the day after the paralysis appeared; definite improvement in the palsy was noted within three days and complete recovery in seven days. The next year, Robbins\textsuperscript{2} reported one case of Bell's palsy in which treatment with cortisone was begun nine days after the onset of the paralysis. Improvement was noted within 24 hours; there was complete recovery in 12 days.

In 1953 Rothendler\textsuperscript{3} reported seven more cases. In all instances, therapy with cortisone was begun from one to ten days after the paralysis appeared. In all but one of the cases, within the first week of treatment recovery had begun and by the end of the second week recovery was complete. In the case that did not respond, the paralysis had been present for ten days and atrophy of the nerve had resulted, as indicated by the faradic test.

The report of five cases by Whitty\textsuperscript{4} lacks details; however, it states that in four cases cortisone therapy was begun within 48 hours and, in the fifth case, within four days of the onset of paralysis. In three of the five cases improvement was noted within 14 days after treatment had been initiated.

Robinson and Moss\textsuperscript{5} reported two cases. In one, treatment with cortisone was started within three days of paralysis; there was definite improvement three days later, and complete recovery in six days. In the other case, the Bell's palsy had been present one week when cortisone was first administered. There was marked improvement by the fifth day of treatment, and by the tenth day there was virtually complete recovery.

In addition to the above cases, there have been two reports of cortisone in the treatment of Bell's palsy that occurred as one manifestation of the Guillain-Barré syndrome. In February 1952, Stillman and Ganong\textsuperscript{6} reported the first case treated with ACTH and cortisone. Among the multiple neurologic signs was bilateral Bell's palsy. ACTH was administered one day after the facial paralysis appeared and within 48 hours the paralysis had disappeared except for a slight defect on the right side of the face. After 12 days of treatment with ACTH, slight facial weakness was again noted. Cortisone was substituted for ACTH, resulting in the prompt disappearance of the facial weakness. The second case was reported by Vernon\textsuperscript{7} in January 1954. In addition to the other neurologic findings, the patient had a bilateral Bell's palsy that had been present for 12 days. On the third day of treatment with ACTH, the patient

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could whistle, wrinkle the forehead, and close the right eye but not the left eye. After ten days of treatment, cortisone was substituted for ACTH. After five days of cortisone therapy, there was only residual damage to the seventh nerve. The patient was maintained on cortisone, and one month after the initial treatment with ACTH, the cranial nerves were found to be intact.

Since January 1954, eight of our patients with Bell's palsy have been treated with cortisone. In five patients the disease was acute; in three it was chronic. We used the cortisone regimen suggested by Robinson and Moss:

- 50 mg. three times a day for two days, followed by
- 50 mg. twice a day for two days, followed by
- 25 mg. three times a day for ten days.

In three patients, the unilateral Bell's palsy was chronic, having been present for, respectively, 2, 10, and 20 months. The patient in whom the disease had been present for two months was possibly slightly worse after the two weeks of treatment. In the patient whose palsy had been present for ten months, there was only slight improvement; no effect was noted in the patient whose disease had been present for 20 months. These three chronic cases are therapeutic failures.

In the five acute cases, cortisone therapy was started from 2 to 14 days after the Bell's palsy appeared. In four of the five patients there was marked improvement within 11 to 17 days after the beginning of treatment. In the fifth patient in whom the cortisone treatment is considered to have failed, there was slight improvement in that he could blow out his cheeks, and smaller quantities of food lodged in the buccal cavity.

CASE REPORTS

Case 1. The patient was a 36 year old white man who, two weeks before initial examination, had noted aching in the left ear and at the angle of the left mandible. The day following this aching, he had numbness of the left side of the face, and found it difficult to close the left eye; also, there was excess lacrimation and blurred vision in that eye. Physical examination revealed a simple type of Bell's palsy with no involvement of the geniculate ganglion or fallopian canal elements. The Bell's phenomenon was not strongly positive; the palsy was most prominent in the perioral musculature. Cortisone therapy was administered for 14 days; three days after treatment was stopped, the patient no longer experienced difficulty chewing food, he could whistle and blow out his cheeks. The only abnormality noted was an exaggeration of the orbicularis oculi-zygomaticus complex when he tried to wink the ipsilateral eye.

Case 2. The patient was a 34 year old white man. He had developed a left facial palsy 23 days prior to, and a right facial palsy two days prior to initial examination. The parasympathetic and taste components of the chorda tympani were not involved. Twelve days after the institution of cortisone therapy, the right (recent) Bell's palsy had almost completely disappeared. There was only a faintly positive Bell's phenomenon on the right; the right corneal reflex was very active; and food no longer collected in the right buccal cavity. The left facial paralysis which had been present for 23 days prior to treatment was not affected. He was advised to continue cortisone 25 mg. twice a day for 12 more days. One month after initiation of therapy, there had been only slight improvement on the left side.

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Case 3. The patient was a 27 year old white man. One week prior to initial examination he had noted that the left side of his face seemed flat, and taste on the left side of the mouth was abnormal. He was unable to blink the left eye, in which there was excess lacrimation. Examination revealed the Bell's phenomenon, contralateral exaggerated ciliary reflex, inability to furrow the left brow, and typical inability to retract the left angle of the mouth. Eleven days after treatment was begun, almost all of the signs had disappeared. The only residual symptoms were incomplete furrowing of the left brow, and weak retraction of the left angle of the mouth.

Case 4. The patient was a 46 year old white woman. Five days prior to initial examination she had noted pain behind the right ear. The following day she noted that liquids escaped from the right angle of the mouth, and she then realized that the right side of her face was paralyzed. Physical examination revealed a simple type of Bell's palsy. Bell's phenomenon was present. Cortisone therapy was started four days after the onset of the palsy. Within three to four days after the onset of treatment, she began to notice improvement. She was seen again two days after the course of treatment was completed. At that time there remained only a slight weakness of the perioral musculature; she could close her eye, furrow her brow, blow out her cheeks, and food no longer collected in the buccal cavity.

Case 5. The patient was a 31 year old white man. Six days prior to initial examination he had noted sagging of the left side of the face, inability to close the left eye, and collection of food in the left buccal cavity. Four days after the onset of symptoms, cervical sympathetic block, performed elsewhere, had no effect. Twenty-two days after the onset of treatment (eight days after treatment was stopped) he again was seen. There was some subjective and objective improvement. He could blow out his cheeks, and food collected in smaller quantities in the left buccal cavity. The Bell's phenomenon remained prominent. He was advised to continue cortisone 25 mg. three times a day for ten more days. At the end of that time, food no longer collected in the buccal cavity, but the other findings were unchanged.

DISCUSSION

Prior to this report, findings in 16 cases of Bell's palsy treated with cortisone had been published. In all of these cases the Bell's palsy had been present no longer than ten days before the initiation of cortisone treatment. In all but three of these patients, there was either marked improvement or cure within 14 days, which appears to be a considerably shorter time than one could expect in the natural course of the disease. In the two reported cases of Guillain-Barré syndrome with facial palsy that were treated with ACTH and cortisone, the effects of therapy were similar.

SUMMARY

The literature of the treatment of Bell's palsy with cortisone is reviewed. Eight additional cases are discussed: three chronic cases and one acute case did not respond to the treatment; four of the five acute cases demonstrated marked improvement within 11 to 17 days after starting cortisone therapy.

The treatment of acute Bell's palsy with cortisone appears to be of value in accelerating recovery from a simple neurapraxic type of injury to the seventh cranial nerve. Although definite conclusions are not justifiable on the basis of
the small number of cases thus far reported, further study of the cortisone treatment is clearly indicated.

References